Table 3. Postoperative Neurologic Complications

Pt. No.	Age (yrs)	Operation	Grade	Modification of Operative Procedures	Aortic Clamp	Post-ECC Evaluation	Symptoms or Signs	Brain CT	Other Risks	Results
1	77	CABG	3	On beating, displaced cannula	Side-biting clamp	New mobile lesion	Right hemiplegia, left hemiparesis	Multiple infarction		Hospital death
2	66	CABG	2	Displaced cannula*	Displaced cross clamp	New mobile lesion	Left hemiparesis, right pyramidal sign, left facial palsy	Multiple infarction (left motor cortex, right occipital)		Alive
3	64	CABG, MAP	3	VF-R	No aortic clamp	New mobile lesion	Chorea	Left caudate head infarction		Hospital death
4	58	CABG	3	VF-R, HCA	No aortic clamp	None	Left hemiparesis	Right frontal lobe infarction (watershed)	Preoperative systemic embolization	Alive
5	74	CABG	3	Displaced cannula	No aortic clamp	None	Weakness of right upper extremity	Left occipital lobe infarction		Alive
6	57	CABG	2	No modification	No modification	None	Right hemiplegia	Left parietal lobe infarction	Postoperative hypotension	Alive
7	70	CABG	1	No modification	No modification	None	Unconsciousness	Multiple infarction		Hospital death
8	62	CABG	1	No modification	No modification	None	Right hemiplegia	Left parietal and temporal lobe infarction (wedge shape)	No other risk	Alive
9	72	CABG	3	VF-R, displaced cannula	No aortic clamp	None	Drowsiness	No apparent change	Multiple infarction	Alive
10	82	CABG	3	VF-R, displaced cannula	No aortic clamp	None	Left hemiplegia	Right occipital lobe infarction		Alive

*Displaced cannula means the arterial cannulation site was changed from the routinely used site to an area of the ascending aorta or arch with less atherosclerosis.
CT = computed tomography; ECC = extracorporeal circulation; HCA = hypothermic circulatory arrest; VF-R = revascularization during ventricular fibrillation; other abbreviations as in Table 1.

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Abbreviations and Acronyms

AAA	= abdominal aortal aneurysm
ASO	= arteriosclerosis obliterans
CABG	= coronary artery bypass graft surgery
CT	= computed tomographic
ECC	= extracorporeal circulation
TEE	= transesophageal echocardiography

in two patients, VSP in 1, myxoma in three and pacemaker lead infection in 1). This study was approved by the institutional Ethics Committee and the Internal Review Board. All patients gave written, informed consent, and all procedures were in accordance with established institutional guidelines.

Techniques. To identify the changes induced by the manipulation, the same sonographic probe was used for two-dimensional epiaortic ultrasonography of the ascending aorta before cannulation (pre-ECC) and after decannulation (post-ECC), as described in a previous study (6). For analytical purposes, the ascending aorta and proximal arch were divided into two segments, designated as the proximal and distal segments. The proximal segment included the aorta from the aortic annulus to 1 cm proximal to the innominate artery, and the rest of the ascending aorta and proximal arch were included in the distal segment. The proximal arch was meticulously dissected to obtain good visualization. This categorization was carried out to relate the proximal segment to the aortic clamp and the distal segment to the aortic cannulation.

In pre-ECC evaluation, we graded the severity of atherosclerosis, taking into account the maximal thickness of the lesion in the proximal and distal segments of the ascending aorta. The severity of the aortic atherosclerosis was assessed as grade 1 if the intima had no thickening or was minimal (<3 mm); grade 2 if the intimal thickening was between 3 and 4 mm; and grade 3 if the thickening was protruding >4 mm, often with an irregular surface or mobile components. Epiaortic ultrasonographic imaging was repeated after decannulation for comparison with data obtained before ECC, using the cannulation site and pulmonary artery as reference points.

All operations were performed under standard cardiopulmonary bypass and moderate hypothermia (28 to 32°C). Arterial cannulations were performed in the ascending aorta (THI angled type aortic 21F or 24F perfusion cannula, Argyle Co., Missouri) or femoral or subclavian artery (William Harvey arterial perfusion cannulae, type 1858, Bardic Co, California), depending on the severity of atherosclerosis. Except for one patient, all anastomoses, including the proximal ones, were performed during a single period of aortic cross-clamping (Fogarty soft-jaw clamp, 86 mm, Baxter Co., California). Because palpation of the ascending aorta underestimates the severity of atherosclerosis, the decision to implement some modifications of the operative technique was made primarily on the basis of the qualitative sonographic information and secondarily on the surgeon's feeling.

Postoperative neurologic events. Patients were examined preoperatively by the physician in charge. If a neurologic deficit was suspected, consultation with the neurologist was sought, but no neuropsychological studies were made preoperatively or postoperatively. Two independent neurologists assessed the development of perioperative stroke. Only permanent or reversible focal complications such as reversible ischemic neurologic deficit or transient ischemic attacks were considered neurologic events. Confusion, agitation, dementia, disorientation or psychosis were considered neurologic events only if new focal neurologic signs were also present. When clinical conditions permitted, computed tomographic (CT) scans of brain were obtained in patients with postoperative neurologic events, and in some patients, plain or angiographic magnetic resonance imaging, carotid duplex scanning and/or scintigraphy of the brain were performed in an effort to seek the cause (embolic or hypoperfusion) of the stroke.

Statistical analysis. To assess the association between the new lesions and the preoperative variables (gender, age, hypertension, diabetes, hyperlipidemia, chronic renal failure, arteriosclerosis obliterans [ASO], abdominal aortal aneurysm [AAA], history of smoking, ischemic heart disease) and the intraoperative variables (duration of aortic cross-clamping, severity of atherosclerosis of the ascending aorta, maximal thickness of the atheroma near the aorta manipulation site, calcification of plaque), univariate analysis was performed.

Univariate analysis was done to assess the association between stroke and history-related variables (gender, age, hypertension, diabetes, hyperlipidemia, cerebral vascular events, chronic renal failure, ASO, AAA, duration of ECC, duration of aortic cross-clamping, severity of atherosclerosis of the ascending aorta, carotid artery disease or presence of new mobile lesions in the aorta).

Univariate testing of variables was performed with the Fisher exact test for discrete variable comparisons. The Mann-Whitney *U* test was used for continuous variable comparisons.

For multiple comparisons, a significance level of 0.003 was set to ensure the whole type I error probability was <0.05, according to the Bonferroni method. All analyses were performed using the SAS Institute version 6.12 software (Cary, North Carolina).

RESULTS

Grade 1 severity of the atherosclerosis was present in 371 patients; grade 2 in 73; and grade 3 in 28. The results of the pre-ECC evaluation indicated that modifications to operative procedures would be required in 63 patients.

Cross-clamping of the ascending aorta was precluded in 23 patients, and in these patients, CABG under hypothermic fibrillation in circulatory arrest or on the beating heart was performed. The arterial cannulation site was changed in 52 patients. Operative modifications were required more often in patients with a severely atherosclerotic aorta ($p < 0.001$).

A new lesion in the ascending aortal intima was identified in 16 (3.4%) of 472 patients after decannulation (Table 1). The echogenicity of new lesions was usually isoechoic to that of the intima nearby, suggesting that the mobile lesions were disrupted plaque or intima. However, owing to a lack of histologic confirmation, there is still a chance of new lesions with thrombus formation on top of the disrupted plaque, so the term "new lesion" was used instead of "new intimal lesion." All lesions except one were distributed in the posterior portion of the aorta. New lesions were of the minor form with some irregularity of the intima in six patients, and of the severe form with mobility or disruption of the intima in 10 patients (Fig. 1 and 2). Six of the severe lesions were attributable to aortic clamping (five to cross-clamping and one to tangential clamping) and the other four to aortic cannulation. Of four new lesions that were thought to be related to aortic cannulation in three patients, the new lesions were located in the direction of the cannula jet and opposite the cannulation side, suggesting that the probable cause was the aortic cannula jet. Three patients in this group had postoperative stroke. No strokes occurred in patients with only new intimal irregularity or intimal tearing with no mobile component.

Univariate analysis was performed to identify the risk factors associated with the new lesions induced by aortic manipulation and with the preoperative and intraoperative variables. Because new lesions caused by aortic cannulation appeared to be multifactorial, these data were not included in the analysis. Thus, only 12 new lesions caused by aortic cross-clamping were analyzed.

In 449 patients who underwent aortic cross-clamping, univariate analysis identified only the maximal thickness of the atheroma near the aorta manipulation site to be a predictor of new lesions (Table 2).

A total of 10 patients (2.1%) sustained neurologic complications. The clinical profiles of these patients with postoperative stroke are summarized in Table 3. Predictors of stroke were determined by univariate analysis (Table 4). Univariate analysis identified ASO, atherosclerosis grade of the ascending aorta and new mobile lesions as predictors of strokes. The preoperative presence of mobile plaque did not influence the occurrence of strokes (Table 4).

Hospital death occurred in six patients (1.3%), three of whom had a stroke and died from infections. The causes of death in the other patients included LOS in one, celiac artery embolization in one and gastrointestinal bleeding in one.

DISCUSSION

Vascular injuries after cardiac surgery have been reported in previous studies (7,8). These include disruption of the intima, laceration, perforation, dissecting aneurysm and false aneurysm. Black et al. (8) reported 50 instances of vascular injuries after cardiac prosthetic valve replacement. Of these, nine patients had injuries caused by surgical clamps. These investigators noted that preexisting abnormalities in the vessel wall indicated a predisposition to certain types of injury. Coelho et al. (9) reported the results of arteriographic and ultrasonic evaluation of vascular clamp injuries, using an in vitro human experimental model. They noted that intimal tears and flaps were observed in 14% and 26% of all atherosclerotic vessels, respectively, whereas clamp trauma is minimal in normal arteries. Ultrasound imaging is superior to arteriography in detecting intimal flaps and can show the majority of intimal flaps caused by vascular clamps.

The dislodgement of material after vascular injury caused by surgical manipulation has been recognized as a major cause of stroke after open heart surgery. Katz et al. (5) observed a mobile atheroma disappear after aortic cannulation during TEE monitoring. The patient had a stroke postoperatively, adding to the evidence that patients with mobile atheromatous disease are at higher risk of embolic strokes during cardiopulmonary bypass. However, there are few reports on the mechanisms that cause intraoperative atheroembolism after cardiac surgery. We performed this study because imaging after decannulation was hypothesized to be helpful in elucidating the mechanism of intraoperative atheroembolism and its associated risk factors. Of 449 patients undergoing open heart surgery with some kind of manipulation of the aorta, a new lesion in the ascending aortal intima was identified in 16 patients (3.6%) after decannulation. Probable causes of new lesions included aortic cannulation, the aortic cannula jet, aortic cross-clamping and tangential clamping.

In a study to determine whether atherosclerotic plaques in the aortic arch are a risk factor in recurrent brain infarction and vascular events, after adjustment for the other risk factors, aortic plaques ≥ 4 mm thick were found to be independent predictors of recurrent brain infarction and all vascular events (10). Using an in vitro human experimental model, Coelho et al. (9) concluded that the occurrence of significant trauma caused by vascular clamps in atherosclerotic arteries is high and occurs whether or not discrete plaques are clamped. In our study, the maximal thickness of the atheroma near the aorta manipulation site was identified as a predictor of new lesions. The incidence of new lesions was 11.8% if the atheroma was ~3 to 4 mm and as high as 33.3% if the atheroma was >4 mm, suggesting that diseased plaque is dangerously fragile. Although no definite conclusion could be drawn owing to the small number of events, these results may help in formulating a strategy for modi-

Table 1. New Lesions

Age (yrs)	Gender	Operation	Grade	Maximal Thickness in Lower Segment (mm)	Ultrasonographic Findings	Location of New Lesions	Probable Cause	Cross-Clamp	CVA
66	M	CABG	2	3.5	New mobile lesion	Posterior	Cross-clamp	Displaced*	Yes
66	M	CABG	2	2.6	New mobile lesion	Posterior	Cross-clamp	Displaced	No
77	M	CABG	3	3.5	New mobile lesion	Posterior	Side-biting clamp		Yes
57	F	CABG	1	1.4	New mobile lesion	Posterior	Cross-clamp	No modification	No
67	M	CABG	2	3.2	New mobile lesion	Posterior	Cross-clamp	Displaced	No
75	F	CABG	2	1.6	New mobile lesion	Posterior	Jet	No modification	No
67	F	CABG, MAP, MVP, TAP	3	4.3	New mobile lesion	Posterior	Jet	Not used	Yes
72	F	CABG	1	2.3	New mobile lesion	Posterior	Jet	Not used	No
50	M	CABG, MAP	2	3.5	Tear in intima	Posterior	Cross-clamp	No modification	No
71	M	CABG	2	1.6	Tear in intima	Anterior	Cannulation	No modification	No
71	F	CABG	2	1.6	Irregularity in intima	Posterior	Cross-clamp	Displaced	No
64	F	AVR	3	1.9	Irregularity in intima	Posterior	Cross-clamp	Displaced	No
65	M	CABG	1	2.2	Irregularity in intima	Posterior	Cross-clamp	No modification	No
66	M	CABG	2	3.9	Irregularity in intima	Posterior	Cross-clamp	Displaced	No
66	M	CABG	2	1.4	Irregularity in intima	Posterior	Cross-clamp	No modification	No
57	M	AVR	3	2.6	Irregularity in intima	Anterior, posterior	Cross-clamp	Displaced	No

*Displaced clamp means the cross-clamp site was changed from the routinely used site to an area of the ascending aorta or arch with less atherosclerosis.
CABG = coronary artery bypass graft surgery; CVA = cerebral vascular accident; MAP = mitral annuloplasty; MVP = mitral valvuloplasty; TAP = tricuspid annuloplasty.

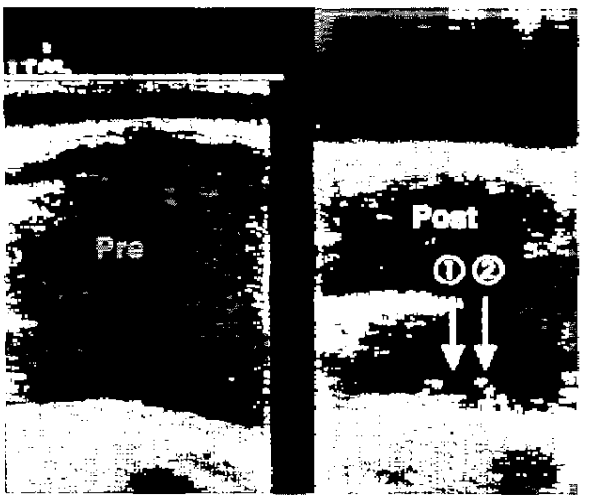

Figure 1. New intimal tear **(arrow 1)** and new mobile lesions **(arrow 2)**, visible at the aortic clamp site, that were not present in the pre-ECC image. Pre = before surgical manipulation.

fication of surgical procedures when dealing with a diseased aorta.

Aranki et al. (11) reported, in a multivariate logistic regression analysis of adverse outcomes in a retrospective study of 310 patients, that use of a partial occluding clamp was a significant predictor of adverse outcome. They noted that application of a partial occluding clamp is probably the most traumatic form of manipulation of the ascending aorta, with the total force of the clamp being concentrated onto a small area, possibly increasing the likelihood of an intimal tear and the dislodgement of plaque or atheromatous material that could embolize on removal of the clamp. At our institution, all anastomoses, including proximal ones, were performed during a single period of aortic cross-clamping in CABG, except in one patient. The only patient in whom a side-biting clamp was used to perform the proximal anastomosis developed new mobile type lesions and had a postoperative stroke. Despite the attempt to clamp a relatively less atherosclerotic anterior portion of the aorta, new mobile type lesions developed in the posterior portion of the same segment. It is highly likely that the application of a side-biting clamp changed the three-dimensional geometry of the aorta, stressing and disrupting the fragile plaque in the posterior wall. Careful application of a side-biting clamp is needed, even if the atherosclerosis involves the posterior part of the aorta. In a diseased aorta, the clamping site of a side-biting clamp, as well as a total cross clamp, should be carefully evaluated.

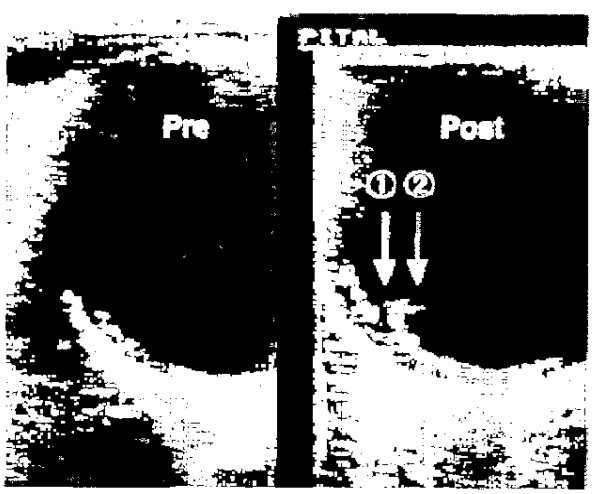

Figure 2. New intimal tear **(arrow 1)** and new mobile lesions **(arrow 2)** likely to have been caused by aortic clamping.

The association between protruding aortic atheromas and spontaneously occurring embolic disease has been reported (12). Tunick et al. (12) reported that protruding atheromas seen by TEE predict future vascular events. Embolic events were more likely to occur when the debris was pedunculated and highly mobile than when it was sessile and immobile. Controversy exists as to whether the mobile component seen by ultrasonography comprises disrupted plaque or thrombus imposed on plaque. Some investigators have reported successful treatment of mobile thrombus by the use of anticoagulant agents or surgical removal (13,14). Histologic examination revealed thrombosis protruding into the aortic lumen, attached to an atherosclerotic lesion (13). However, in our hospital, histologic examination of surgically removed mobile lesions revealed atheromatous plaque with cholesterol crystal and macrophage invasion (not yet reported). In our study, the echogenicity of new lesions was usually isoechoic to that of the intima, suggesting that mobile lesions comprised disrupted plaque or intima. Taking these facts into account, and considering the relatively short time lag between aortic manipulation and reevaluation by echocardiography (10 to 15 min after decannulation), it is likely that, in our cases, the mobile component seen on the post-ECC images was not thrombus but was disrupted plaque.

In the present study, new mobile lesions created by surgical manipulation have been identified as predictors of strokes, in addition to the more familiar predictors, ASO and atherosclerosis of the ascending aorta, described in previous studies (1,6). However, no direct cause–effect relation between stroke and the new lesions was proved. It is not known whether mobile lesions indeed embolized. However, by univariate analysis, new mobile lesions were identified as risk factors of strokes. This suggests that new lesions could play a role in triggering strokes with or without preexisting atherosclerotic disease. This may result from the atheroembolism of mobile plaque or thromboembolism derived from newly disrupted lesions in the early or late postoperative stage.

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Table 2. Univariate Analysis for New Lesions

Variables	Post-ECC Irregularity	Mobile Lesions or Laceration	p Value
Male gender	1.4% (4/281)	1.8% (5/281)	0.5477
Age (years)			0.4779
<50	0% (0/37)	0% (0/37)	
50 to 59	1.4% (1/74)	2.7% (2/74)	
60 to 69	2.4% (4/166)	1.8% (3/166)	
70 to 79	0.7% (1/151)	0.7% (1/151)	
>80	0% (0/21)	0% (0/21)	
Maximal L (mm)			<0.0001
<3	0.5% (2/389)	0.3% (1/389)	
3 to 4	3.9% (2/51)	7.8% (4/51)	
>4	22.2% (2/9)	11.1% (1/9)	
Calcification	2.9% (1/35)	0% (0/35)	>0.9999
Ischemic heart disease	1.2% (4/325)	1.8% (6/325)	0.5244
Ischemic heart disease and valvular disease	0% (0/31)	3.2% (1/31)	0.5809
Aorta cross-clamp time (min)			0.4595
<60	1.6% (2/127)	2.4% (3/127)	
60 to 120	1.4% (4/290)	0.7% (2/290)	
>120	0% (0/32)	3.1% (1/32)	
Dialysis patients	5.3% (1/19)	0% (0/19)	0.4087
Renal failure (creatinine >2.0 mg/dl)	3.1% (1/32)	0% (0/32)	0.5929
AAA	11.1% (1/9)	0% (0/9)	0.2181
ASO	3.8% (1/26)	11.5% (3/26)	0.0032
Hypertension	1.2% (3/256)	1.6% (4/256)	>0.9999
Hyperlipidemia	1.6% (2/125)	0.8% (1/125)	>0.9999
Diabetes mellitus	2.7% (3/110)	0.9% (1/110)	0.4989
Total	1.3% (6/449)	1.3% (6/449)	

AAA = abdominal aortal aneurysm; ASO = arteriosclerosis obliterans; ECC = extracorporeal circulation.

Although not all new lesions result in strokes, efforts not to create new lesions, including suitable strategies based on pre-ECC imaging, careful maneuvers and meticulous feedback by means of post-ECC imaging, may reduce the number of strokes.

Study limitations. This nonexperimental study was undertaken to clarify the causative mechanism of embolic stroke during open heart surgery. For obvious ethical reasons, data could not be obtained on the impact of surgical manipulation on the severely diseased aorta in which the risk of embolization was high. In addition, because efforts were made to avoid creating atherosclerotic lesions during manipulation, the frequency of new lesions was decreased, so the number of new lesions was too small to draw any definite conclusions.

Previous studies have demonstrated that the presence of triglyceride-rich lipoproteins may predict a plaque type particularly vulnerable to rupture, because the echolucency of carotid atherosclerotic plaques on computerized ultrasound B-mode images has been associated with a high incidence of brain infarcts as identified by CT scans (15). However, because our study was retrospective, plaque echogenicity was not included in this analysis. Measurement of echogenicity be means of computerized ultrasound B-mode images may enable more accurate identification of vulnerable plaque to be made in the future, before surgical manipulation.

Because intraoperative TEE was not used routinely in this series, TEE data were not included in this analysis. However, because we meticulously dissected the proximal arch before pre-ECC ultrasonography, we believe that inclusion of this data may reflect, to some extent, the degree of atherosclerosis of the arch. Continuous monitoring by TEE, combined with pre-ECC and post-ECC imaging by means of epiaortic ultrasonography, may help in clarifying the mechanism by which strokes are caused.

Conclusions. This study demonstrated the association between new lesions created by surgical maneuvers and postoperative strokes. Embolic strokes were more likely to occur if new lesions were complicated by intimal disruptions, especially of the mobile type. If thick plaque (especially >4 mm) is noted near the manipulation site, modifications in surgical procedures will be necessary to avoid creating new lesions.

Table 3. Postoperative Neurologic Complications

Pt. No.	Age (yrs)	Operation	Grade	Modification of Operative Procedures	Aortic Clamp	Post-ECC Evaluation	Symptoms or Signs	Brain CT	Other Risks	Results
1	77	CABG	3	On beating, displaced cannula	Side-biting clamp	New mobile lesion	Right hemiplegia, left hemiparesis	Multiple infarction		Hospital death
2	66	CABG	2	Displaced cannula*	Displaced cross clamp	New mobile lesion	Left hemiparesis, right pyramidal sign, left facial palsy	Multiple infarction (left motor cortex, right occipital)		Alive
3	64	CABG, MAP	3	VF-R	No aortic clamp	New mobile lesion	Chorea movement, left hemiparesis	Left caudate head infarction		Hospital death
4	58	CABG	3	VF-R, HCA	No aortic clamp	None	Left hemiparesis	Right frontal lobe infarction (watershed)	Preoperative systemic embolization	Alive
5	74	CABG	3	Displaced cannula	No aortic clamp	None	Weakness of right upper extremity	Left occipital lobe infarction		Alive
6	57	CABG	2	No modification	No modification	None	Right hemiplegia	Left parietal lobe infarction	Postoperative hypotension	Alive
7	70	CABG	1	No modification	No modification	None	Unconsciousness	Multiple infarction		Hospital death
8	62	CABG	1	No modification	No modification	None	Right hemiplegia	Left parietal and temporal lobe infarction (wedge shape)	No other risk	Alive
9	72	CABG	3	VF-R, displaced cannula	No aortic clamp	None	Drowsiness	No apparent change	Multiple infarction	Alive
10	82	CABG	3	VF-R, displaced cannula	No aortic clamp	None	Left hemiplegia	Right occipital lobe infarction		Alive

*Displaced cannula means the arterial cannulation site was changed from the routinely used site to an area of the ascending aorta or arch with less atherosclerosis.
CT = computed tomography; ECC = extracorporeal circulation; HCA = hypothermic circulatory arrest; VF-R = revascularization during ventricular fibrillation; other abbreviations as in Table 1.

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Table 4. Univariate Analysis for Strokes

Variables	Incidence of Strokes	p Value
Male gender	3.0% (9/299)	0.202
Age (yrs)		0.4356
<50	0% (0/37)	
50 to 59	2.6% (2/77)	
60 to 69	1.7% (3/177)	
70 to 79	2.5% (4/159)	
>80	4.5% (1/22)	
Ischemic heart disease	2.9% (10/347)	0.138
Ischemic heart disease and valvular disease	3.1% (1/32)	0.508
Dialysis patients	0% (0/21)	1.0
Renal failure (creatinine >2.0 mg/dl)	11.1% (4/36)	0.008
AAA	9.1% (1/11)	0.424
ASO	16.1% (5/31)	< 0.001
Hypertension	3.6% (10/277)	0.014
Hyperlipidemia	4.4% (6/135)	0.072
Diabetes mellitus	2.6% (3/116)	0.713
Mobile atheroma	25.0% (2/8)	0.022
Aorta grade		< 0.0001
1	0.5% (2/371)	
2	2.7% (2/73)	
3	21.4% (6/28)	
New mobile lesions	33.3% (3/9)	< 0.001
Bruit	0% (0/11)	1.0
Postoperative hypotension	14.3% (1/7)	0.280
Aorta cross-clamp time (min)		0.0111
<60	4.1% (6/148)	
60 to 120	1.4% (4/290)	
>120	0% (0/32)	
ECC time (min)		0.4897
<90	0.6% (1/166)	
90 to 150	3.1% (8/258)	
>150	2.2% (1/46)	
History of CVA	4.3% (3/69)	0.336
Total	2.1% (10/472)	

CVA = cerebral vascular accident; other abbreviations as in Table 2.

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